

Deaths in long-term paced patients

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The survival rate in 649 patients with chronic atrioventricular block with pacing is compared with that in 113 unpaced patients and with that in the population of England and Wales matched for age and sex. The causes of the 145 deaths occurring in the paced patients are analysed. Thirteen were attributed to pacing. There were also 33 late sudden deaths. Of the 82 necropsies, 62 included detailed examination of the coronary arteries and serial sectioning of the conducting tissue. The pathology underlying the atrioventricular block was ischaemic disease in 20 per cent, and in 8 of the 62 necropsies there was evidence of a terminal ischaemic incident.

Full analysis of the incidence of sudden unexplained death shows it to be no more common when fixed rate pacemakers compete with inherent beats than in the absence of competition.

In 13 years (1960/1972), 771 patients have been paced on a long-term basis at St. George's Hospital, all but 27 for complete atrioventricular block. Since 1962 our standard method has been with an implanted pacemaker and a transvenous endocardial electrode. This paper reports the survival rate of patients with complete block paced by this method and the causes of the deaths.

Of the total series, 649 were paced by an implant with a transvenous endocardial electrode. Some of these also underwent a period of pacing with myocardial electrodes placed on the heart at operation and some were paced for periods with an endocardial electrode with the lead brought through the skin to an external pacemaker. Such patients are included in the survival statistics but only the deaths occurring while pacing with an implant and transvenous electrode are analysed here. The series included 375 men and 274 women. The age distribution is shown on the histogram (Fig. 1). Of the pacemakers used in this series, 93 per cent were manufactured by Devices Ltd. The remainder covers a wide variety of pacemakers made in the U.S.A., Sweden, Holland, and West Germany.

Survival comparisons

The survival curve is seen in Fig. 2, which also indicates the survival of 113 unpaced patients with complete atrioventricular block, and the expectation of life of the population of England and Wales matched with the paced series for age and sex (latest available figures 1961). Our series of 113

unpaced patients was collected before artificial pacing was introduced and in the early days of pacing (1960 to 1965) when pacing was relatively unreliable. These unpaced patients include all patients with complete block seen in the department over this period and their mean age and sex distribution does not differ grossly from the paced series. They must, however, be considered as a selected

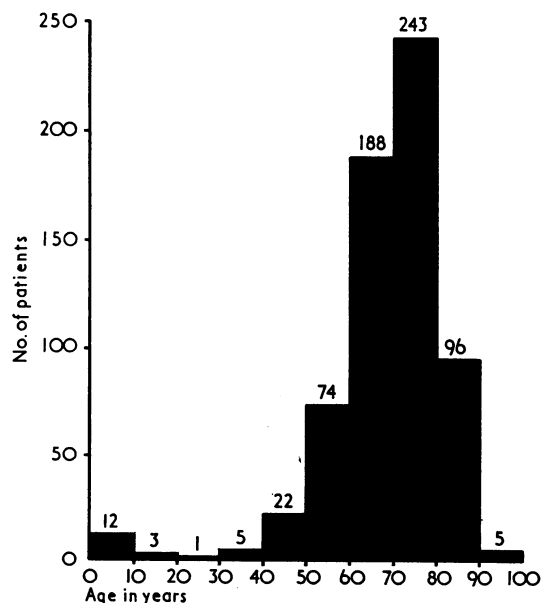


FIG. 1 Age distribution of 649 paced patients.

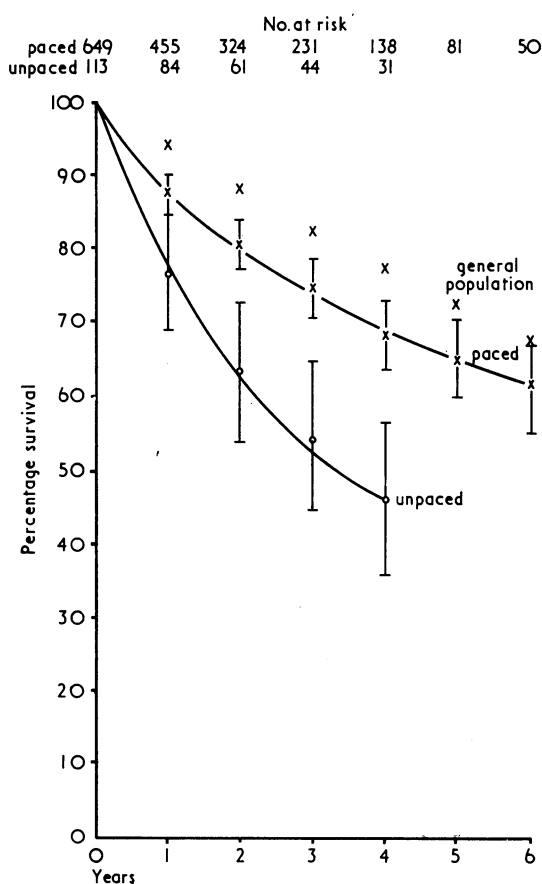


FIG. 2 Survival of paced and unpaced patients with complete atrioventricular block. The unlinked curve shows the survival rate of the general population matched with the paced patients for age and sex. The vertical lines indicate the 5 per cent confidence levels (standard deviation $\times 2$).

group not strictly comparable with those who were paced (see discussion below).

Analysis of deaths

Previous reports from this hospital have shown how difficult it is in life to determine the aetiology of atrioventricular block (Harris *et al.*, 1969). Standard necropsy procedures rarely provide definite evidence of aetiology of atrioventricular block nor of the mechanism of death. We have obtained necropsy reports in 81 of the 145 deaths. Moreover, we have been particularly fortunate in 62 of these cases in having had an unusually detailed study of the heart, involving postmortem coronary angiograms and the

study of serial sections of the conducting tissue. This has shown in every case the pathological process causing the atrioventricular block though in many its aetiology remains unknown. In determining the ultimate mechanism of death the description of the terminal clinical picture is often more enlightening than necropsy and an attempt has been made to obtain full details in every case. A careful personal assessment of all the information, both clinical and when available from necropsy, has allowed classification into the following groups.

Group 1. 46 patients – ‘Various unrelated’. Cause of death known and not related to underlying cardiac pathology nor to pacing.

Group 2. 49 patients – ‘Cardiovascular’. Cause of death known to be cardiovascular and often related to the underlying cardiac pathology, but not to the block or pacing.

Group 3. 13 patients – ‘Attributable to pacing’. Cause known to be related to pacing or its failure.

Group 4. 33 patients – ‘Late sudden’. Later deaths either unheralded or in 7 patients occurring hours after an incident which could have been a failure to pace, a myocardial infarct, etc.

Group 5. 4 patients – ‘Insufficient information for classification’.

These groups are shown in Table 1 which also shows in which patients death occurred within 4 weeks of a pacing procedure, in which a necropsy was obtained and in which serial sections of the conducting tissue were studied.

Group 1: various unrelated

Of the 46 deaths, 8 occurred within 4 weeks of starting pacing, but death was not considered to be related to pacing. It had been started in 4 to cover surgical treatment for unrelated conditions which were the cause of death: 2 of these 8 early deaths were caused by pneumonia. One was caused by a cerebrovascular accident in a hypertensive patient 11 days after a pacemaker change. The remaining death within 4 weeks was because of an acute abdominal condition, which was not operated on as the patient was 90. There was no necropsy.

The 22 necropsies revealed a cause of death which was not cardiovascular in each case. Of the 46 deaths, 15 were caused by malignant disease.

Group 2: cardiovascular

The causes of death in these 49 patients can be classified as follows: progressive cardiac failure, 26; acute myocardial infarction, 9; cerebrovascular accident, 8; and arrhythmia unrelated to pacing, 2; and one each to systemic embolism, pulmonary

TABLE I *All deaths*

Group		No. of deaths	No. within 4 weeks of pacing	Necropsy	Serial sections of conducting tissue
1	Various unrelated	46	8	22	14
2	Cardiovascular	49	5	33	26
3	Attributable to pacing	13	5	13	10
4	Late sudden	33	0	14	12
5	Not classifiable	4	0	0	0
Total		145	18	82	62

embolism, intracardiac and intravascular thromboses, and aortic aneurysm.

The progressive cardiac failure subgroup included a few in whom renal failure played a prominent part. Necropsies were obtained in all 9 of the acute myocardial infarctions. In 4 of them death occurred within 4 weeks of a pacing procedure: in one this was a routine change of pacemaker: in the other 3, necropsy evidence suggested that the infarct had preceded the implantation of the pacemaker, though the clinical features had suggested a long standing block. In each of these 3 artificial pacing was beneficial, but had the acuteness of the condition been appreciated, pacing would have been carried out with an external unit avoiding, at least for a time, the procedure of implantation.

In only 3 of the 8 patients dying with a diagnosis of cerebrovascular accident was a necropsy obtained. It was not possible to determine how many of these might be attributed to embolism.

Both the patients who were observed to die in a tachyarrhythmia had been noted to have had similar arrhythmias on many occasions. Demand pacing and suppressant drugs for over two years in each had prevented syncope, but not the paroxysms of tachycardia. One patient was aged 29 and did not come to necropsy. Necropsy showed a cardiomyopathy in the other.

Group 3: attributable to pacing

These 13 patients can be subdivided as follows: failure to pace, 6; speeding pacemaker, 3; septicaemia, 3; early unexplained sudden, 1.

Necropsy was obtained in all: 5 occurred within 4 weeks of a pacing procedure. Of the 6 caused by a failure of pacing, 2 were shown to be attributed to defective pacemakers. In the other 4 the pacemaker output was tested and found to be normal and the fault presumably lay in the contact between electrode

and heart or in the electrode wire. The cessation of pacing in 2 of these patients resulted in intractable arrhythmias which were never controlled.

The mechanism of death in the 3 with speeding pacemakers is likely to have been by ventricular fibrillation. It is possible that the normally working pacemaker precipitated ventricular fibrillation in the single case of this group classed as an early unexplained sudden death, but ventricular fibrillation was not recorded though the patient was being monitored with an oscilloscope. This patient died on the second day of pacing and necropsy revealed calcific involvement of the conducting tissue. The possibility that a normally working pacemaker induced ventricular fibrillation causing some of the later deaths is discussed below.

In 3, death was caused by septicaemia and these require fuller annotation.

MM, female, aged 58: In the third year of pacing, 6 months after an implant, the wire ulcerated through the skin at the vein entry site. She was not referred to hospital and 4 months later died of staphylococcal septicaemia.

ES, female, aged 54: Complete atrioventricular block followed an aortic Starr-Edwards valve replacement. Syncopal attacks occurred and were not relieved by apparently satisfactory pacing with a demand pacemaker. Ventricular tachycardia was observed to be the cause of syncope; ventricular fibrillation then followed and was successfully treated by defibrillation. The pacemaker was taken out and the ventricular tachycardia controlled by fast pacing. During this period sepsis was seen to develop along the wire track. When it was thought to be controlled and the arrhythmia had apparently settled a pacemaker was again implanted. Eighteen days later she died from staphylococcal septicaemia.

RH, female, aged 2. A series of epileptiform attacks led to various neurological deficits and the diagnosis of complete heart block. Pacing with myocardial electrodes was started at the age of 23 months, but

failed and was replaced by endocardial pacing. Three months later *Streptococcus viridans* septicaemia developed and despite treatment proved fatal in a few weeks. Necropsy revealed endocardial fibroelastosis.

The septicaemia occurred in the first 2 of these cases as a result of the wire being exposed close to the site of vein entry allowing staphylococci to enter the blood stream. The cause in the third case of the child with streptococcal septicaemia cannot be explained in this simple mechanical manner.

Group 4: late sudden

Necropsy was obtained in 14 of these 33 deaths. It is, however, in this group of patients that necropsy often reveals no explanation, and it is possible, but by no means certain, that pacing may have failed, resulting in cardiac arrest, or that ventricular fibrillation without a period of asystole may have been the fatal mechanism.

In 7 of the 33 deaths in this group death was preceded by a sudden onset of symptoms. In 3 of these cases coming to necropsy there was no evidence of an acute ischaemic event and the pacemaker on testing was faultless. In one of them there was doubtful postmortem evidence of failure of the electrode system. In this patient and the other 6 with premonitory symptoms before death there was no evidence of a slow pulse suggesting failure to pace.

Pulmonary embolism

Massive pulmonary embolism was considered the cause of death in only one and minor embolism was found in only 2 others of the 81 necropsies.

Acute myocardial infarction

The presence of a recent occlusive lesion in the coronary system is difficult to demonstrate without a much more thorough and time-consuming necropsy technique than is usually adopted. In 8 of the 62 detailed necropsies undertaken in this series there was evidence to show this as the mode of death. The detailed study of the conducting system showed that ischaemic heart disease was the underlying cause of the atrioventricular block in 20 per cent of these 62 necropsies. In some, death resulted from coronary occlusive disease, though it was not the underlying cause of the atrioventricular block. *Pacemaker-induced fatal arrhythmia* must be assumed to have occurred in the 3 with speeding pacemakers (group 3). In only one patient have we evidence of terminal ventricular fibrillation following a paced rhythm with a normally functioning pacemaker. This was one of the patients referred to above in group 2, whose pacemaker was implanted

shortly after an acute ischaemic episode. The pacemaker was a demand model. In 3 other patients dying of acute myocardial infarction in hospital the terminal rhythm was monitored. In one ventricular tachycardia but not ventricular fibrillation was observed. The other 2 died of cardiogenic shock; arrhythmia was not a prominent feature of the terminal events in these 2.

Ventricular fibrillation induced by a normally functioning pacemaker cannot be excluded in many of the patients dying unmonitored. This could have been the mechanism of death in all 37 patients in groups 4 and 5 and in 3 patients in group 2, and in one referred to above in group 3 (i.e. 41 patients).

Competition between artificial and spontaneous rhythms

Over the past few years we have used demand pacing for patients in whom we anticipated competition, but it was only in 1969 that these non-competitive pacemakers became readily available to us, and before that time we had accumulated considerable experience in fixed rate pacing of patients with competition. Since April 1970 we have kept traced electrocardiograms or written records of the oscilloscopic appearances in paced patients at intervals of about 4 months. These make it possible to separate the patients paced since that date into those without competition and those with competition. The latter can be subdivided into those who have been shown on at least one occasion at rest to have competition from (a) sinus rhythm, (b) ectopic ventricular beats, or (c) both sinus rhythm and ectopic ventricular beats.

Table 2 shows the incidence of unexplained death in these groups when paced with fixed rate and with demand pacemakers. Only 12 of the deaths considered as possibly caused by iatrogenic arrhythmias occurred after adequate records were being kept (April 1970). The significance of these figures is considered below. The salient points are that there were 6 deaths with fixed-rate pacing in 377 patients (mean period 2.4 years), as compared to 6 deaths with demand pacing in 258 patients (mean period 1.4 years). Competition with fixed rate pacing was observed in 168 (mean period 2.4 years) with 4 deaths, as opposed to no competition in 455 patients (mean period 1.9 years), with 8 deaths.

Discussion

Unpaced survival

The survival curve of the unpaced chronic atrioventricular block patients requires some comment. Many of the patients were observed after the intro-

TABLE 2 *Deaths with and without competition with fixed rate and demand pacing*

	Fixed rate			Demand		
	No. of patients	Patient-years of pacing	Deaths	No. of patients	Patient-years of pacing	Deaths
No competition observed	209	479	2	67	76	3
Competition observed	168	411	4	191	287	3
(a) SR	56	125	1	117	177	1
(b) V	71	182	2	42	59	2
(c) SR+V	41	104	1	32	51	0
Total	377	890	6	258	363	6

Deaths include only those in which iatrogenic arrhythmia from a normally functioning pacemaker cannot be excluded (see text).

All electrocardiogram observations made at rest.

Competition (a) Patients with observed sinus competition (SR), but no ectopic ventricular competition (V).

(b) Patients with observed V but not SR.

(c) Patients in whom both forms of competition were observed.

TABLE 3 *Survival with complete atrioventricular block without pacing*

Authors	No. of patients		
Johansson (1966)	119	Acute infarcts and digitalis blocks excluded	60 per cent survived 1 year
Penton, Miller, and Levine (1956)	104	Included 21 acute infarcts	26.2 months mean survival
Curd <i>et al.</i> (1961)	130	High proportion of children	47 per cent survived 2 years
Friedberg <i>et al.</i> (1964)	100	20 lost to follow-up.	30 survived 1 year
Pader and Levy (1966)	73	Includes about 15 per cent acute infarcts	45 per cent survived 2 years
Campbell (1944)	50		Mean survival of those dying, 2.5 years; author calculates overall expectation of life at 4.6 years
Cosby <i>et al.</i> (1965)	36	Excluding acute infarcts	55 per cent survived 1 year

duction of long-term pacing in 1960, but were not initially paced for a variety of reasons. In the early years of pacing it was felt that if syncopal attacks were infrequent conservative management was justified. Of the 113 patients, 78 had had syncopal attacks. Two patients were considered too senile to be paced and 2 refused pacing. The mean age of the 113 unpaced patients was 67 and two-thirds were men, a similar proportion to the paced patients.

The survival of 78 per cent of the unpaced patients at 1 year and the mean survival between 3 and 4 years shows a much better prognosis than the few published series. These are summarized on Table 3. The mean survival is not specifically stated in most of these reports but it can be concluded that it lies between 1 and 2 years. Johansson's report (1966) which provides the firmest evidence, gives a 60 per cent survival at 1 year.

Comparability of paced and unpaced

Johansson has shown that the mortality in complete block is concentrated to a period shortly after its onset. It is necessary, therefore, to consider whether there is a bias between the unpaced group and the paced group resulting from the inclusion of this danger period more frequently in one group than in the other. It is often difficult to determine from the patient's history exactly when the block first occurred. Patients who have had syncopal attacks, however, are usually able to state when the first attack occurred. The clinical history showed that the first attack had been within 6 months of the first attendance at hospital in 32 per cent of the unpaced, and within 6 months of starting pacing in 50 per cent of those paced.

Both groups must be regarded as selected from the total population of patients with block, in that

they represent the survivors of the initial danger period, but the fact that a greater proportion of the paced patients had less than a 6-month history of syncope implies that this paced group would, if not paced, have had a poorer survival rate than the unpaced group.

Survival curves

There is a remarkable similarity of survival of paced patients with that of the general population matched for age and sex. After the first year the two curves on Fig. 2 run almost exactly parallel.

Pulmonary embolism

Many of the reported series of transvenously paced patients include one or two instances of death from pulmonary embolism. The highest incidence was reported by Bernstein, Rotem, and Peretz (1971) who found pulmonary embolism in 9 of their 22 deaths occurring in the first year of pacing. It had been massive in 4 of them. In our limited experience of pacing with myocardial electrodes (77 patients), we had a higher incidence than in our transvenous series, in which only one massive pulmonary embolus occurred.

Acute myocardial infarction

There are surprisingly few necropsy reports of more than one or two cases of paced patients dying of coronary thrombosis. However, Robboy *et al.* (1969) recorded 7 necropsies, in 3 of which acute myocardial infarction was considered the cause of death. Donmoyer, DeSanctis, and Austen (1967) reported 17 deaths, 4 of which were attributed to myocardial infarction with necropsy confirmation in 3. Edhag's (1969) series of 52 necropsies included 17 recent myocardial infarctions. These figures come from the U.S.A. and Sweden, countries in which the incidence of acute myocardial infarction may be higher than in this country. The increased incidence is, however, not sufficient to explain the finding in our series that only 8 cases out of 62 with detailed necropsies died of acute myocardial infarction.

Early sudden deaths

The argument that the risk of sudden death is greater with fixed rate pacing is often based on Bilitch's reports (1969) on *early* sudden death in his 86 patients. He recorded fixed rate pacing in 40 patients, 8 dying in hospital: 6 of the 8 deaths were among the 20 who showed competition. By comparison in his 46 with triggered pacing only 2 died after operation. Both deaths occurred in the 30

who showed competition. Of the 10 deaths, 6 were documented as occurring in ventricular fibrillation.

Our 4-week mortality from known ventricular fibrillation, acute infarction, or unexplained sudden death has been 5 in 649 patients. (This excludes two which resulted from speeding of the pacemaker.) Of the 5 deaths, 4 were attributable to acute myocardial infarction (failure of output rather than arrhythmia was the mode of death in 2 of them); in the fifth the terminal event was not documented.

Others commenting on Bilitch's results have pointed out that the postoperative mortality of 10 in 86 (8 in 40 fixed rate) is much higher than anything recorded elsewhere and that the patients cannot, therefore, be considered comparable to most chronic heart block pacing practice. The 6 cases documented as dying in ventricular fibrillation may have passed through a stage of asystole; the recording of ventricular fibrillation terminally does not prove that it has been directly precipitated by the pacemaker.

Later sudden deaths

The 33 deaths in this category, represents 23 per cent of all the deaths, which is in keeping with some reported large series (Scheppekot *et al.*, 1969; Fauchier *et al.*, 1971) though greater than that reported by others (Davidson *et al.*, 1972; Ducloux *et al.*, 1971; Lagergren and Karlöf, 1968; Chardack *et al.*, 1969) who gave a proportion of 14 to 20 per cent of all deaths.

Before interpreting the experience displayed in Table 2 with regard to the differing risk of sudden death with fixed rate as opposed to demand pacing, it is essential to consider what differences there were in the groups of patients paced by the two methods. Much of the experience shown in Table 2 covers a period before demand pacemakers became available to us. However, when demand pacemakers were available, the choice of pacemaker was based on the likelihood of competition, patients in whom complete atrioventricular block was known to be intermittent being more often paced by the demand unit. This introduced an element of selection into the groups paced with the two types of pacemaker.

Is the expectation of life, regardless of pacing, different in intermittent complete atrioventricular block from that in permanent block? The evidence on this point with patients without pacing is limited and varied. Most authors find that intermittent complete block in unpaced patients has a prognosis for life little different from established complete block (Friedberg, Donoso, and Stein, 1964; Hollingsworth *et al.*, 1969; Penido, Cotton, and Cosby, 1965; Lau *et al.*, 1965). However, Johansson's

(1966) figures show that intermittency of block is associated with a significantly better survival at 1 year (64% of 70 with intermittent v 30% of 94 with established block). Hayase *et al.* (1967) also reported it as a favourable feature.

As Johansson's and Hayase's reports are the only 2 of the 6 which suggest that the expectation of life is better in intermittent as opposed to permanent block, no firm conclusions can be drawn. But when comparing the results of treatment of patients with intermittent block with patients with permanent block it is necessary to bear in mind the possible different prognosis in the two groups.

Our figures in Table 2 show that 56 patients with sinus competition, but not ventricular ectopic competition, were paced with fixed rate pacemakers (mean period of pacing 2.2 years), with 1 death. This can be compared with 209 patients without known competition paced with fixed rate pacemakers (mean period of pacing 2.3 years), with 2 deaths. The 56 patients with sinus competition and fixed rate pacing can also be contrasted with 117 patients who showed return of sinus rhythm, but were paced with demand pacemakers. There was again 1 death (mean period of pacing 1.5 years). The figures do not suggest any increased risk of sudden death when there is competition with sinus rhythm.

We have felt that competition from beats of ventricular origin may be of more serious import than conducted beats. We know of no clear evidence on this point. The figures with fixed rate pacing provide a comparison between 2 deaths in 71 patients with ectopic competition (mean period of pacing 2.6 years) and 2 deaths in 209 patients with no competition (mean period of pacing 2.3 years). The 2 deaths in 71 patients can also be compared with 2 deaths in 42 patients with similar competition but paced by demand pacemakers (mean 1.4 years).

It is often stated that there is an increased risk of sudden death in chronic atrioventricular block patients paced with fixed rate pacemakers, if either conducted rhythm returns or ventricular ectopic competition occurs. We suggest that our figures do not support this view. The numbers are insufficient to provide statistical proof but are large enough for one to anticipate that some indication of the increased risk would show. Most but not all the published evidence on this matter suggests that the risk may be real. But is it sufficient to be used as an argument for the use of non-competitive pacemakers in these chronic atrioventricular block patients?

The following reports provide some information on this point. Scheppokat *et al.* (1969) reported 10 sudden deaths in 250 fixed rate as opposed to 0 in 124 demand. Parsonnet *et al.* (1968) reported 9

unexplained deaths, in 8 of which there had been competition. Fauchier *et al.* (1971) published collective figures showing 5 per cent sudden deaths in 2527 fixed rate as opposed to 1.5 per cent in 993 non-competitive pacers. Ducloux *et al.* (1971) in another collective analysis, which included Bilitch's figures on early deaths, attributes death to ventricular fibrillation in 3.2 per cent of 1502 fixed rate compared with 0.9 per cent in 675 non-competitive. Contrary experience comes from Furman and Escher (1970) who record 15 sudden deaths in 150 patients over 7 years, none of which had competitive rhythms. They have also stated that inexplicable sudden deaths occur with equal frequency with asynchronous (fixed rate) and non-competitive pacing. These reports though of significant numbers of paced patients do not unfortunately state for what length of time pacing was carried out, though the importance of this factor has been mentioned by several authors.

There is a complicating factor which probably influences the results of all these reports, including our own. Experience with fixed rate pacing generally predates that with non-competitive pacing. Techniques can be assumed to have improved so that one would expect a lower death rate with more recent experience and this should favour the results with demand pacemakers. This may be partially offset by the apparently poorer outlook of patients being accepted over the last few years. The average age of patients coming to pacing was increasing year by year and the proportion with a short history of syncope was also increasing.

The output of the pacemakers is a further factor which could influence the incidence of pacemaker induced ventricular fibrillation. We have favoured relatively low output pacemakers partly because their life is likely to be longer, but also because we recognized the magnitude of the artificial stimulus as a factor in the ability of a pacemaker to produce ventricular fibrillation.

The fibrillation threshold can be lowered by many factors which include the irritation of the pericardium which inevitably follows any operation to place electrodes on to the heart's surface. The early reports of pacemaker induced ventricular fibrillation (Sowton, 1965; Siddons and Sowton, 1967) were based on experience with epicardial pacing, and any risk with that technique cannot be assumed to apply with the transvenous pacing. In order to eliminate this factor, this report and the discussion have been confined to transvenous pacing.

We conclude that the often assumed risk of sudden death from fixed rate pacing competing with sinus rhythm in patients with intermittent chronic atrioventricular block has not been established by

published experience, and that this report by failing to show any increased risk adds some evidence to the view that the risk of sudden death should not be used as an argument for demand pacing in these patients. The published material includes even less evidence to show whether competition from heart beats of ventricular origin may carry a different risk from that of conducted beats. We suggest that it might more often be associated with sudden death, but our numbers are insufficient to allow a conclusion.

Choice of fixed rate or demand pacemakers

In spite of our conclusion that the risk of sudden death from sinus competition is negligible, we do not always use fixed rate pacemakers. There are other considerations when selecting the type of pacemaker for an individual patient. When there is sinus rhythm and a demand pacemaker is used, the haemodynamic benefit of correctly timed atrial contraction benefits the patient. Unfortunately, it is not possible to forecast which of the patients apparently in established atrioventricular block may revert after pacing to sinus rhythm. For this reason it is often recommended that the first implant should be a demand unit. When it comes to changing the unit the choice is easier, as it is possible to look back over our records. If no competition or only occasional ventricular ectopic beats have been observed, we used a fixed rate unit. The advantages of the fixed rate Devices pacemakers over their demand model is a longer life and reduced cost. With the aid of oscilloscopic testing (Siddons and Davies, 1973; Davies and Siddons, 1973) we were able to get between 2½ and 3 years of life out of the fixed rate units as opposed to only 2 years from the demand unit, in which it is more difficult to forecast battery depletion. Currently the Devices demand unit costs 67 per cent more than the fixed rate.

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